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Metacognition as a Predictor of Change in the Treatment for Borderline Personality Disorder: A
Preliminary Pilot Study

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Abstract

There is evidence that a key feature in Borderline Personality Disorder (BPD) is the difficulty to reflect on one's own as well as on other people's state of mind. So far, no study has examined the link between metacognition and symptom change presented by BPD patients across treatment. This preliminary pilot study represents a first step in describing impairments in different dimensions of metacognition and their connection with the symptoms presented by a $N = 10$ BPD sample. The transcripts from the first and the penultimate session of a ten-session version of Good Psychiatric Management were analysed. The global score and the three subscales of the Metacognition Assessment Scale-Revised (MAS-R) were used in order to assess metacognitive abilities: Understanding of one's own Mind, Understanding of Other's Mind, and Mastery. Symptoms were assessed with the OQ-45. Results show a general low level of metacognition. Therapy sessions with BPD patients seem to be characterized by impairments in metacognition, notably in the reflection on other's states of mind and in the use of this information to solve conflicts and problems. Moreover, sessions highlighting a higher level of the Understanding of Other's Mind at the onset of treatment were linked with better outcome compared to sessions showing poorer metacognitive abilities in this area. Implications for an integrated treatment for BPD and future research are discussed.

Key-Words: metacognition, borderline personality disorder, psychotherapy, outcome

The understanding and treatment of BPD requires investigating core mechanisms of pathology, which can then be the target of intervention across different psychotherapeutic perspectives. The past decades saw the emergence of many studies focusing on the impairments of BPD patients to reflect upon their states of mind and those of others (Bateman & Fonagy, 2004; Fischer-Kern, Buchheim, Hörz et al., 2010; Fischer-Kern, Doering, Taubner et al., 2015; Fonagy, 1991; Gullestad, Johansen, Høglend et al., 2013; Katznelson, 2014; Kernberg, Diamond, Yeomans et al., 2008; Levy et al., 2006; Semerari, Carcione, Dimaggio et al., 2005). This set of abilities has been defined with different, often overlapping terms (Choi-Kain & Gunderson, 2008; Dimaggio & Brüne, 2016) such as “social cognition” (Fiske & Haslam, 1996), “theory of mind” (Brüne, 2005), “mentalization” (Bateman & Fonagy, 2004) or “metacognition” (Semerari et al., 2003). Throughout this study, the term metacognition will be used as it is consistent with the tradition underlying the instrument used here to analyse this ability. Difficulties in understanding mental states range from poor awareness of one’s own feelings, difficulties in inferring the thoughts and feelings underlying others’ behaviours, and the inability to connect different mental states in an integrated narrative. Indeed, poor skills in emotional recognition and reflection can be at the root of many aspects of BPD pathology, such as emotional dysregulation, impulsivity, self-harm and suicidality (Bateman & Fonagy, 2004). Social conflicts, poor sense of identity and poor quality of relationships can also stem from difficulties in understanding both one’s own and others’ mental states and using this knowledge to foster prosocial communication.

Difficulties in understanding mental states are believed to be both a treatment target and a possible predictor of change in psychotherapy, and therefore need to be included in an integrated treatment for personality disorders (Livesley, Dimaggio, & Clarkin, 2016). On the one hand, good outcome therapies are supposed to foster patients’ skills to understand mental states and to

use this information to cope with stressors, and thus to live a more adapted life (Bateman & Fonagy, 2010). On the other hand, people with metacognitive difficulties can have more problems in understanding the therapeutic process and fruitfully using the contents of the therapy exchange (Gullestad et al., 2013). It is therefore to be expected that good metacognition at treatment onset predicts better outcome, and also that good outcome treatments yield changes in the ability to understand mental states.

In this paper we first describe the definition of metacognition and the different mentalistic impairments present in BPD. We then review evidence for the possible role of metacognition in the psychotherapeutic process of BPD. We finally frame research questions about metacognition in a short-term treatment for BPD.

Metacognition: Definitions and assessment

Metacognition includes a range of capacities human beings use to identify mental states, both in oneself and others, reason about them, forming complex and integrated representations of oneself and others, and use mentalistic knowledge for problem solving (Carcione, Dimaggio, Conti et al., 2010; Semerari, Carcione, Dimaggio et al., 2007). A certain overlap exists between metacognition and mentalization, defined by Bateman and Fonagy (2004, p.21) as “the mental process by which an individual implicitly and explicitly interprets the actions of himself or herself and others as meaningful on the basis of intentional mental states such as personal desires, needs, feelings, beliefs, and reasons”. Mentalization has been operationalized with the Reflective Function Scale (RFS; Fonagy, Target, Steele & Steele, 1998) which aims to assess the quality of mentalization and involves different dimensions, as those concerning the self and others, implicit and explicit modes of functioning and cognitive and affective mentalizing. The RFS provides a single and global score.

Several differences can be highlighted concerning mentalization and metacognition. First, unlike mentalization, metacognition does not assume that the activation of the attachment system is the only source of disrupted abilities (Dimaggio, Semerari, Carcione et al., 2007).

Furthermore, metacognition, when operationalized with the Metacognition Assessment Scale-Revised (MAS-R; Carcione et al., 2010), evaluates separately different processes that can be disturbed. For example, the MAS-R has a specific subscale dedicated to the ability to use mentalistic information for purposeful problem solving, which allows to disentangle its unique role in psychopathology and psychotherapy (Carcione et al., 2011; Outcalt et al., 2016).

Metacognition includes three broad functions. *Understanding of one's own Mind* includes 3 subfunctions: a) *Monitoring*, or the recognition and definition of cognitions and emotional states and the link between them and the behaviour. It is close to the concept of alexithymia, which includes the difficulties in identifying feelings and describing them to other people as well as a concrete and externally oriented style of thinking (Taylor, Bagby, & Parker, 1997); b) *Differentiation* is the ability to make a distinction between representations and reality, as thoughts are not objective descriptions of reality. A good level of differentiation would be “*I thought that my entire life was a failure but then I realized it was only a thought and that good things happened to me too. It made me feel better about myself*”; c) *Integration* relates to the construction of an integrated view of the self despite the variations in life events.

The second function is the *Understanding of Other's Mind*. It includes: a) *Monitoring* as the ability to identify what others are thinking and feeling and the connections between thoughts, affects and overt behaviour; b) *Decentration* refers to the ability to imagine that other people have their own perspective which is likely different from our own: “*In his place, I would have*

badly reacted to the waiter's rudeness, thinking it was shameful to be treated like that. He stayed quite calm and polite, maybe thinking the waiter had had a bad day."

Mastery includes three levels of metacognitive reflection for the purpose of soothing suffering and solving interpersonal difficulties. The first level concerns strategies implying a limited use of information on mental states in order to solve problems, such as reducing emotional arousal by acting directly on the body (e.g. physical exercise). Dysfunctional strategies at this level include the use of drugs or self-harm. Second level strategies include the ability to purposefully alter one's behaviour, for example inhibiting one's action tendency, and re-focusing attention (e.g. deciding to stop worrying). Finally, the third level demands a greater metacognitive effort, such as taking a critical distance from thoughts or beliefs underpinning a problem, as well as using knowledge about the mental states of others to face interpersonal struggles.

Metacognition in Borderline Personality Disorder

Poor metacognition has been suggested to be an aspect of core pathology of personality disorders (Semerari et al., 2007; 2014; 2015). Concerning BPD, it has been argued that metacognition is overall low, and in particular that patients struggle with distancing themselves from their own firmly held beliefs and with forming an integrated representation of self and others (Bateman & Fonagy, 2004; Livesley, 2003).

Regarding the knowledge and understanding of one's own state of mind in BPD, many studies have focused on poor emotional awareness, also labelled alexithymia (Taylor et al., 1997). Poor affect awareness seems to be present in BPD (Levine, Marziali, & Hood, 1997; McMain et al., 2013; Joyce et al., 2013; New et al., 2012), though other studies have not found such a link (Nicolò et al., 2011). When analysing metacognition in patients' discourse in both

psychotherapy sessions and semi-structured interviews, Semerari and colleagues (2005; 2014; 2015) did not find poor awareness of one's own affects and thoughts.

Concerning the understanding of others' state of mind, recent results suggest that patients presenting with BPD have difficulties in forming complex ideas about others and in seeing the world from the perspective of the others. These difficulties are made obvious when laboratory tasks are complex enough, mirror real-life conditions, or elicit distress (Brüne, Walden, Edel, & Dimaggio, 2016; New et al., 2012; Petersen, Brakoulias, & Langdon, 2016; Preissler et al., 2010) or when analysing patients' discourse in semi-structured interviews (Dimaggio et al., 2009; Dinger et al., 2014; Outcalt et al., 2016; Semerari et al., 2005; 2014; 2015).

The ability to regulate emotions is considered as a key problem in BPD and emotional dysregulation stems in part from poor emotional awareness (Linehan, 1993). BPD patients often use dysfunctional, nonmentalistic strategies (Bateman & Fonagy, 2004) in order to regulate unpleasant emotional states and psychological suffering, such as self-injury or substance abuse (Linehan, 1993; Scott, Stepp, & Pilkonis, 2014). This can be described as poor metacognitive mastery and can be demonstrated by low levels on the Mastery subscale of the MAS-R (Semerari et al., 2007). Mastery has been found impaired in BPD patients (Carcione et al., 2011; Outcalt et al., 2015).

BPD has been linked with difficulties in different areas of metacognition. Results are still inconsistent regarding the specific mentalistic impairments in BPD patients and further studies are needed to get a better understanding of them. In the light of the possible role of metacognition in BPD pathology, researchers focused on its evolution through psychotherapy and its impact on therapy outcome.

Can metacognition change through psychotherapy?

Most psychotherapies for BPD are designed to improve patients' mentalistic capacities (Bateman & Fonagy, 2004; Dimaggio, Montano, Popolo, & Salvatore, 2015) or to work in order to promote awareness of one's own psychological functioning which likely foster abilities to reflect on mental states, such as the Good Psychiatric Management (GPM; Gunderson & Links, 2014) adopted in the present study. In their 1-year RCT, Levy et al. (2006) compared the change in the RFS in patients in intensive psychotherapeutic treatments. The patients allocated to Transference-Focused Therapy (Kernberg, 1993) showed a significant increase in mentalizing while this improvement was not found in patients under Dialectical Behaviour Therapy (Linehan, 1993). Fischer-Kern and colleagues (2015) replicated the findings and found a unique improvement in the RFS in the sample treated with Transference-Focused Therapy compared to the control group treated by experienced community therapists. Nevertheless, it should be noted that other studies did not demonstrate such a linear change in RFS, notably in a hospitalization-based psychodynamic treatment for PDs (Vermote et al., 2010; 2011).

Studies using the MAS showed a trend in terms of improvement in metacognition through Cognitive-Behavioural Therapy and Metacognitive Interpersonal Therapy (Dimaggio et al., 2007) in case studies of various PDs (Dimaggio et al., 2009; Dimaggio, Procacci, et al., 2007; Semerari et al., 2003).

Is metacognition a predictor of outcome?

The idea that poor metacognition in BPD at therapy onset predicts outcome has yielded mixed findings. Alexithymia, measured with the Toronto Alexithymia Scale (TAS-20; Bagby, Parker, & Taylor, 1994), has been pointed out as an outcome predictor (Ogrodniczuk, Piper, & Joyce, 2011) but Joyce and colleagues (2013) did not replicate this finding, which might have

been due to clinicians' increased capacities to deal early on with patients' difficulties in describing their emotions, which could have reduced the toxic effect of alexithymia.

Gullestad et al. (2013) found a moderator effect of RFS: patients with low RFS underwent a greater improvement in psychosocial functioning in an outpatient treatment. No difference was found between two treatment conditions for patients with medium or high levels of RF. Nevertheless, the study did not find a significant predictor effect of RFS on outcome. A longer 6-year follow-up of the same sample (Antonsen et al., 2016) found that patients with lower baseline RF had better outcomes in outpatient individual therapy as compared to patients with higher RF who achieved better outcomes in a group therapy based step-down treatment program.

Overall, metacognition is impaired in BPD and results are inconsistent in terms of metacognitive improvement during successful therapy. Metacognition, or related constructs as alexithymia and mentalizing, could be predictors of symptom change, but here again results are mixed. In light of inconsistencies from previous studies and given the lack of studies where metacognition as operationalized here was measured, to date we have no findings speaking for whether metacognition improves during psychotherapy for BPD and predicts outcome. Moreover, no study has yet examined the link between metacognition and symptom change in BPD patients in a short-term treatment.

The present study

This preliminary pilot study is the first application of the MAS-R to a BPD sample and a first step in the exploration of the links between metacognition and symptom change in this population. An underlying goal is also to test the feasibility of this research design for a larger study, as MAS-R scorings require intensive training and time to be done in a methodical way.

This is the reason why we used a small convenience sample treated with a short version of the GPM (Gunderson & Links, 2014) from a previously published study (Kramer et al., 2014).

Before attending to the hypothesised link between metacognition and symptom change, two preliminary research questions are explored:

1. What is the level of metacognition before treatment? Though normative data for MAS-R do not yet exist, we expect low range scores of metacognition (e.g. < 2.5), which correspond to less than adaptive levels of metacognitive abilities.
2. Does metacognition improve through short-term treatment? At this level, this hypothesis is exploratory. It may well be that either metacognition does not improve after ten sessions because it is not a treatment target in the approach adopted, or that it increases as GPM focuses among other elements on interpersonal functioning and its ties to the clinical symptoms.
3. Finally, is there an association between the level of metacognition at intake and symptom change presented by BPD patients between the first and penultimate sessions of treatment? As the main hypothesis, we predict a positive link between metacognitive capacities at intake and symptom change during treatment.

Method

Participants

A total of $N = 10$ patients were selected from a previously published study (Kramer et al., 2014). In the latter, a total of 74 BPD patients were included and assigned either to a short-version of the GPM treatment or to the same treatment where the Motive-Oriented Therapeutic Relationship (MOTR; Caspar, 2007), a form of therapeutic relationship based on an individualized case formulation, was added. Patients of the present study were randomly selected

from the GPM arm of the Kramer et al. (2014) study. We used the control sample and the basic treatment to test in a more rigorous manner our hypothesis of a link between metacognition and symptom change in the context of a minimum treatment intervention. A random selection was made, and comparison between our sample and the larger sample of Kramer et al. (2014) was tested with a naked eye assessment. Criteria were similar concerning the age, gender, fulfilled BPD criteria, employment and marital status. However, our sample reported more symptoms on the OQ-45 at intake (mean = 108.50, $SD = 23.43$) compared to the larger sample (mean = 95.00, $SD = 27.00$). Moreover, a higher rate of patients in the larger sample were using medication (60%) compared to our sample (20%).

Our participants were 9 women and 1 man, all French-speaking, with a mean age of 33 years ($SD = 4$; median = 32.50, $IQR = 24$). 7 patients were single, and 3 were married. 7 were unemployed and 3 were part-time working. Only 2 individuals had medication. Table 1 provides an overview of the main data and scores obtained by our patients at intake and at the end of the treatment.

All patients were recruited from an outpatient university psychiatry clinic and provided written consent. Inclusion criteria were the following: being between 18 and 65 years old and having a SCID-II (First & Gibbon, 2004) confirmed diagnosis of BPD. Patients had on average 7 ($SD = 0.4$; median = 7, $IQR = 2.25$) fulfilled BPD criteria on the SCID-II. Exclusion criteria were: existence of a psychotic disorder, mental retardation and substance abuse in the foreground. Comorbid psychiatric disorders are shown in Table 1.

Therapists

Five therapists were in charge of the treatment of the 10 patients included in our study: 1 therapist treated 1 patient, 3 therapists treated 2 patients and 1 therapist treated 3 patients. All

therapists were working at the outpatient clinic where the patients were recruited for the study. They were psychiatrists and psychologists with at least 1 year of psychiatry residency and a basic psychodynamic background. Therapists were trained at the onset and during the study in the empirically validated GPM and the treatments were supervised twice during the whole process. Supervisors had training in psychodynamic therapy and had received a specific training in psychiatric management of BPD patients conforming to the GPM model (Gunderson & Links, 2014).

Treatment

The patients received a 10-session treatment which is a short version of the GPM treatment for BPD patients. The elaborated treatment has the following goals: to establish psychiatric diagnoses, comorbidities, and psychiatric anamnesis; to define the main topics and the treatment target; to identify short-term objectives; to name and deal with difficulties interfering with the treatment; and finally to formulate the relational interpretations of core conflictual themes (Kramer et al., 2014). The GPM model was integrated as a useful first-line treatment and as a preparation for a long-term psychotherapy (Kolly et al., 2016).

Treatment fidelity was assessed using the General Psychiatric Management Adherence Scale (Kolla et al., 2009) and showed high treatment integrity (Kramer et al., 2014). Each assessment, as well as the adherence observer ratings, was generally made by 1 research assistant with the help of 3 other research assistants when needed.

Instruments

Comorbid psychiatric disorders

Comorbid psychiatric disorders on axis I and II were assessed by two structured interviews: the Mini International Neuropsychiatric Interview (Lecrubier et al., 1997) and the SCID-II (First & Gibbon, 2004).

The Outcome Questionnaire-45.2 (OQ-45; Lambert, Morton, Hatfield, et al., 2004) aims to assess mental health functioning and its evolution during psychotherapy. The items are assessed on a 4-point Likert scale, ranging from 1 (never) to 4 (always). A global score is then calculated. The clinical cut-off for the global score is 63. It has been translated and validated in French (Emond et al., unpubl. data). The scale was given after the first and penultimate sessions. It was filled in between sessions, at home by the patients. Cronbach's alpha for the total score was $\alpha = 0.94$.

The Metacognition Assessment Scale-Revised (MAS-R; Carcione et al., 2010) detects metacognitive change in individual's narratives and was originally developed for psychotherapy transcripts and specialized interviews. It provides a global score of metacognition and a score for three subscales with several subfunctions:

- 1) Understanding of one's own Mind (UM subscale) measures the ability of a person to think about its own mental states. It includes *Monitoring*, *Differentiation* and *Integration* subfunctions previously described (see Introduction).
- 2) Understanding of Other's Mind (UOM subscale) assesses the ability to think about the mental states of others. It includes the *Monitoring* of others' mental states and the *Decentration* subfunctions.
- 3) Mastery (M subscale) is the representation of mental states involving suffering or psychological conflicts and the adoption of an active attitude to use mentalistic information

to soothe suffering and solve conflicts. It includes three different levels, from a more behavioural to a more metacognitive level.

For each subfunction, ratings are made on a 5-point scale from 1 = “scarce” (sporadic, poorly articulated, not spontaneous, probing does not generate improvement) to 5 = “sophisticated” (sustained talk about mental states, descriptions are rich, talk of mental states is spontaneous or there is an autonomous elaboration of a question/suggestion). In case a subfunction does not occur in the interview, the MAS-R gives the possibility to score it as “Not Engaged”.

Lysaker et al. (2007) found significant correlations among the three subscales of the MAS-A, the previous version of the MAS-R. Because of our small sample size, we did not perform such correlational analyses here. They will be carried out on a larger sample in a future study.

MAS-R assessment and rating

For each patient, the first and penultimate sessions were tape-recorded and transcribed (Mergenthaler & Stigler, 1997). After dividing the transcripts into scoring units, two independent raters, the first and the second authors, scored them with the MAS-R. Both were blind to any details regarding the sample and the sessions. The second author was one of the creators of the MAS-R and the first author was trained by the latter until good reliability was achieved. A consensus rating was used for the data. On the 20 sessions coded, two were used for training purposes, so inter-reliability in assessing the metacognitive subfunctions was not calculated for them. Inter-rater reliability was therefore calculated for 90% of the transcripts ($N = 18$) with Intra-Class Coefficients (Shrout & Fleiss, 1979). It was good to excellent, with a mean $ICC(2,1) = .85$ ($SD = .01$, range = .70 - .91).

Statistical analyses

Kolmogorov-Smirnov and Levene's tests were used to assess the normality and homogeneity of variance of the data and help to select the proper statistical analyses. An evaluation of the total score and subfunction scores of metacognition were provided using item-mean scores. We then used the Wilcoxon signed-rank test to compare differences between scores in MAS-R at intake and after ten sessions. Finally, Spearman correlations were used to determine the link between metacognition and symptoms as measured through the OQ-45. For this, we subtracted the OQ-45 score after ten sessions from the score obtained at onset in order to get the delta value. Given the exploratory nature of the study, no Bonferroni correction for the number of tests was made.

Results

Preliminary analyses

Metacognition scores. Table 2 presents the scores obtained by participants at each subscale at intake and at the end of the treatment. Our sample showed a MAS-R mean score of 1.77 ($SD = 0.36$; median = 1.58, $IQR = 1.48$) at onset, and a MAS-R mean score of 1.93 ($SD = 0.48$; median = 1.86, $IQR = 0.83$).

Metacognitive change in a 10-session treatment. Regarding the evolution of MAS-R scores between the first and the penultimate session, even if an improvement is observed, the differences were not significant, except for the first-level Mastery strategies ($z = 1.96$, $p = .05$; Table 2).

Links between metacognition and symptoms. For our main hypothesis, we first tested if patients improved in terms of symptoms. They showed a mean symptom level of 108.5 ($SD = 7.4$), median = 113.50 ($IQR = 4$, range = 71-144) on the total OQ-45 at intake and a mean of 77.4

($SD = 10.2$), median = 77 ($IQR = 42$, range = 19-130) after 9 sessions, this difference being significant ($z = -2.4$, $p < .05$; $ES = 3.49$).

Then, Spearman correlations revealed a link between the metacognitive level at intake and symptom change (Table 3). More precisely, the total MAS-R score at the beginning of treatment tended to be linked with symptom change during treatment measured by the OQ-45 delta ($r_s = -.52$, $p = .06$). Focusing on the three subscales of MAS-R, the UM and M subscales did not show links with the change on the OQ-45. In contrast, we found a relationship between the UOM subscale and symptom reduction ($r_s = -.56$, $p < .05$).

Clinical examples

To illustrate the metacognitive impairments our patients suffer from, clinical examples are presented below. They are verbatims from the session transcripts. Patient 3 is a 35-year-old female who had a low score in the UM scale at onset (mean = 1.69). She's talking about her mood swings:

Therapist: *So, you always relapse. Can you describe to me how this happens?*

Patient: *It's feels like a depression, but it happens suddenly, it is not progressive. Suddenly I feel bad, I don't have anymore desires, I want to die, to call for help, to do drugs...*

T: *That's it.*

P: *But I don't feel the emotional changes coming up, I feel bad only 2-3 days or 2-3 weeks before.*

T: *And at that moment, what is going on?*

P: *It depends on the degree of sadness or on external events in fact. Sometimes I can call for help*

and say “now I need medicine because something’s wrong”, or it becomes bigger and bigger and I say to myself “anyway there is no point to call for help, that won’t fix the problem, my partner is not able to understand my suffering” and I hurt myself.

In this example, she describes a lack of differentiation. She does not take a critical distance from her own beliefs and thoughts (“*anyway there is no point to call for help, that won’t fix the problem, my partner is not able to understand my suffering*”) are considered as being reality. Moreover, we can observe a low level of Mastery here (use of self-harm). A better differentiation ability is illustrated by Patient 10 (mean UM at onset = 3.17), a 55-year-old man talking about a situation that triggered disappointment:

T: *How did that make you feel?*

P: *Mmmh... disappointment, I felt disappointed and irritated. And even though I consider myself to be a flexible person... perhaps I’m not. Perhaps I consider myself to be flexible and in reality I’m not.*

In his reflective process, he can take a critical distance from his thoughts, to the point of considering that they may not necessarily mirror reality.

Discussion

This preliminary pilot study aimed to investigate several questions regarding metacognition in therapy and its connection with symptom change within a sample of patients presenting with BPD.

We expected that metacognitive abilities were in low range, and the scores obtained by patients seem to confirm this. In relation to previous research, we found that the present sample

had equal levels of metacognition compared to samples of psychotic patients (MacBeth et al., 2014; Mitchell et al., 2012). This is in line with conceptual, clinical and research work showing that metacognitive abilities in BPD patients can be severely impaired (Bateman & Fonagy, 2004; Kernberg, 1993; Semerari et al., 2005). As the psychometric properties of this scale are not formally established yet, our study makes a first contribution to how severe the metacognitive deficits in BPD could be. It also gives an idea about their nature, with impairments in every dimension and very prominent problems in the use of mentalistic information in order to cope with life challenges (mastery).

Second, we also aimed to explore the evolution of metacognition through a 10-session treatment. Even if a trend towards a positive evolution was shown in the majority of metacognitive dimensions, the difference between the first and penultimate sessions was only significant for the first-level Mastery strategies. This result has to be taken with caution because of the sample size and the multiple testing carried out. Moreover, even if the difference reached the significance threshold, we are not able to conclude that a clinical change occurred, as the MAS-R score at the end is still under a moderate level of metacognition. A significant clinical change may occur with a long-term treatment or a treatment focused on metacognition. However, as a basis for hypotheses for future studies, several comments can be made: strategies which use a limited amount of mentalistic knowledge in order to solve problems, e.g. understanding one is distressed and undertaking physical exercise in order to calm oneself down, require a low reflective ability. This could partially explain the quick improvement of this subfunction compared to the others for which long-term treatment is necessary (Bateman & Fonagy, 2009; Carcione et al., 2011; Levy et al., 2006). It is likely that either because of their growing sense of trust in the therapist or because they were offered simple instructions on how to

cope with suffering, patients learned to identify distress and tolerate it. More complex abilities such as calming oneself down thanks to a more comprehensive view of oneself and others did not develop. This is to be expected because treatment was very short. Research is needed to see whether the GPM is able to promote metacognition when applied for longer periods and, more generally, whether different treatments are able or fail to promote metacognition.

Finally, we assumed a link between early levels of metacognitive skills and symptom change. The global level of metacognition at intake seems to be linked with symptom decrease during treatment, even if this trend did not reach statistical significance. A link was found for intake sessions showing a greater ability to grasp the others' states of mind and reflect on them. Sessions characterized by higher level in this domain at the beginning of treatment seem to be connected with greater outcome. Again, as this exploratory study included a small sample size, there is an urgent need for replication in a larger sample and results are to be considered preliminary at best. Nevertheless, a possible explanation for this result, if replicated, is that with reduced capacity to understand the mental states of other people at the beginning of the therapy, patients have more difficulty understanding the therapist's perspective and the use of therapy. Thus, they will have problems in adopting new perspectives coming from the therapeutic exchange and using those to deal effectively with social challenges in the short term. It is also possible that with reduced understanding of the state of mind of others, BPD patients feel more alone when suffering and deprived of social support. Nevertheless, it is possible that these metacognitive abilities were moderated by the therapist's techniques.

The indication of a possible relation between metacognition and symptoms underlines the necessity to take into consideration the abilities of patients to recognize and understand mental states of other individuals, including the therapist, as a common principle of change in therapy

for BPD. A hypothesis could be that early in treatment, the level of metacognition reveals something about the potential evolution of the patient's symptoms throughout treatment. In this case, it would be possible for the clinician to assess and detect early difficulties in metacognition and then deliver the appropriate intervention according to the patient's capacities and with an attempt at improving them progressively as prescribed in Mentalization-Based Treatment (Bateman & Fonagy, 2004) or in Metacognitive Interpersonal Therapy (Dimaggio, Montano, Popolo, & Salvatore, 2015). Indeed, both symptoms and interpersonal functioning have to be prime targets in therapeutic treatments for PD patients (Bateman & Fonagy, 2004; Linehan, 1993; Livesley, 2003; Livesley et al., 2016).

This preliminary pilot study also shows the importance of taking into account separate dimensions of metacognition. Indeed, if we only had considered the total MAS-R score, precious information would have been lost, such as the role and predictive value of the ability to reflect upon other's states of mind on symptom change. These results are in line with a conceptualisation of metacognition as a complex and multi-factorial concept (Semerari et al., 2003, 2005, 2007).

A number of limitations need to be highlighted in this preliminary pilot study. First of all, it involved a small convenience sample which makes it difficult to generalize and limits the statistical power of the results, making the results tentative at best. As an example, we can cite the high effect sizes found (Table 2), which are probably not robust in such a small sample. Moreover, our sample had a very high female prevalence (9/10), which also contributes to the low generalizability of the results. Furthermore, the sample showed a metacognition level comparable to the one obtained by psychotic patients. As this present study is the first to use the MAS-R in a BPD sample, no data comparison yet exists. It is therefore difficult to estimate if our

sample is representative of BPD patients in terms of metacognition levels. For all these reasons, future studies should include a control/comparison group in order to make firm conclusions about the specificity of metacognitive change. It is still conceivable that different therapeutic or relational techniques can have an impact on metacognitive processes (Semerari et al., 2003). Kramer, Caspar, and Drapeau (2013), for example, showed the impact of the MOTR (Caspar, 2007) on decreasing cognitive biases. It is possible that patients treated in the other arm of the trial (Kramer et al., 2014) from which our sample is taken, where the MOTR was used, could have showed a different pattern of prediction of metacognition over symptoms and of metacognitive change. Another limitation is that the transcripts come from therapy sessions and patients' metacognitive abilities do not only depend on the patients themselves, but also strongly on the therapist's ability to work consistently with the patient's metacognitive level. Therefore, there is the need to replicate the finding with patients treated by different therapists coming from different therapeutic approaches, in order to explore whether addressing metacognitive problems can be considered part of an integrated treatment for BPD. A further limit is that our main outcome, the symptom level, was self-reported by patients; therefore, the need for observer-rated measures of change remains. It was also administrated in a non-standard manner (after and not immediately prior to session). Finally, short-term treatment results were analyzed and do not inform us more about long-term effects, especially considering that metacognitive skills require time to grow and exert their beneficial effects on symptoms and social functioning (Carcione et al., 2011; Dimaggio et al., 2009; Levy et al., 2006; Semerari et al., 2005).

Irrespectively of their therapeutic orientation, clinicians can focus on problems in the different metacognitive domains and adjust their response to the patients' current abilities to make sense of mental states, taking into account the heterogeneity in BPD patients (Bateman &

Fonagy, 2004; Leiman & Stiles, 2001; Levy & Scala, 2015; Ribeiro et al., 2013; Semerari et al., 2007). For example, a combination of first-line interventions aimed at promoting basic mastery strategies, which would produce good outcomes, can be coupled with interventions aimed at improving basic self-reflective skills, such as awareness of one's own mental processes. In later stages, the focus would move on to promoting the understanding and the use of more complex mental states.

In summary, our preliminary results are in line with previous studies showing strong impairments in mentalistic abilities in psychotherapy with patients presenting with BPD. The use of the MAS-R could allow to draw a more precise map of the impairments in BPD metacognitive abilities as well as to quantify the impact of these on symptom change during short-term treatments. This preliminary study highlighted the need for further research: first, confirming the role of patients' metacognitive abilities in the early sessions of treatment on symptom evolution; second, exploring the impact of the therapist and the therapeutic relationship on metacognition. Such studies are currently in progress.

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Table 1

Characteristics of the ten patients

Subjects	Age	Gender	GAF at intake	Comorbidities on axis I *	MAS-R total scores		OQ-45 total scores	
					Intake	Penultimate session	Intake	Penultimate session
1	30	Female	50	1	1.54	1.58	144	130
2	19	Female	55	3	1.52	1.76	71	84
3	35	Female	60	1	1.62	2.00	77	45
4	22	Female	60	1, 6	1.92	1.96	125	61
5	26	Female	65	2	1.84	2.54	131	118
6	20	Female	60	1, 3	1.52	2.67	116	71
7	44	Female	50	1, 4	2.52	1.60	112	77
8	48	Female	60	1, 5	1.56	1.74	89	77
9	37	Female	60	7	2.24	2.38	115	19
10	55	Male	60	2, 6	1.46	1.12	105	92

* 1 = depression, 2 = dysthymia, 3 = anorexia, 4 = bulimia, 5 = panic disorder, 6 = alcohol abuse, 7 = intelligence limitation

Table 2

Means of MAS-R scores obtained by patients at intake and after ten sessions

MAS-R scales and subscales	Intake Mean (<i>SD</i>) Median (<i>IQR</i>)	Penultimate session Mean (<i>SD</i>) Median (<i>IQR</i>)	z	p	Effect size
MAS-R total	1.77 (.36) 1.58 (.48)	1.93 (.48) 1.86 (.83)	1.27	.20	-.37
Understanding of one's own Mind (UM)	2.24 (.16) 2.05 (.82)	2.37 (.18) 2.41 (.85)	.66	.51	-.76
Monitoring	2.80 (.65) 2.72 (.11)	2.75 (.70) 2.88 (.86)	.66	.51	.07
Differentiation	1.62 (.55) 1.41 (.83)	1.88 (.55) 1.66 (.71)	1.13	.26	-.47
Integration	1.42 (.51) 1.20 (.81)	1.88 (.69) 1.66 (1.26)	1.25	.21	-.75
Understanding of Other's Mind (UOM)	1.49 (.13) 1.47 (.83)	1.56 (.14) 1.47 (.87)	.56	.58	-.52
Monitoring	1.58 (.45) 1.66 (.86)	1.63 (.46) 1.60 (.82)	.56	.58	-.11
Decentration	1.31 (.38) 1.17 (.63)	1.37 (.48) 1 (1)	.41	.68	-.14
Mastery (M)	1.58 (.11) 1.58 (.64)	1.88 (.17) 1.89 (.64)	1.38	.17	-2.06
Basic requirements	1.80 (.63) 1.83 (1.17)	2.20 (.59) 2.33 (1)	1.50	.14	-.66
1st level strategies	1.41 (.30) 1.45 (.39)	1.94 (.62) 2 (1.21)	1.96	.05	-1.06

2nd level strategies	1.75 (.49)	1.76 (.60)	-.95	.34	-.02
	1.75 (.83)	1.5 (.75)			
3rd level strategies	1.69 (.21)	1.86 (.62)	.74	.46	-.36
	1.67 (.67)	2 (.93)			

Table 3

Correlations between metacognition at intake and symptom change (delta OQ-45)

	r_s	p
MAS-R Total	-.52	.06
Understanding of one's own Mind (UM)	-.40	.12
Understanding of Other's Mind (UOM)	-.56	.04
Mastery (M)	-.43	.10

All correlations are Spearman's rho.